

Echocardiographic findings after acute carbon monoxide poisoning¹

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Myocardial lesions are frequently seen at necropsy after fatal carbon monoxide poisoning. Clinically, while there have been numerous reports of chest pain and electrocardiographic changes associated with acute carbon monoxide poisoning, other evidence for left ventricular abnormality has not been reported. The echocardiographic findings in five cases of non-fatal poisoning are presented here. Abnormal left ventricular wall motion was shown by echocardiography in 3 cases. Motion returned to normal in 2 of the 3 in follow-up tracings. Echocardiograms on 3 of the 5 patients showed mitral valve prolapse. Though the mitral valve prolapse may have been present before the poisoning, the reported high incidence of papillary muscle lesions in fatal cases suggests a possible relation of the prolapse to the effects of the carbon monoxide poisoning.

Myocardial damage, including necrosis, is commonly seen at necropsy after fatal carbon monoxide poisoning (Marek and Piejko, 1972; Gey, 1924; Ehrich, Bellet, and Lewey, 1944; Beck, Schulze, and Suter, 1940). It has also been shown in experimental animals who were sacrificed after non-fatal poisoning (Ehrich *et al.*, 1944). The subendocardial and papillary muscle areas of the left ventricle are most frequently involved (Marek and Piejko, 1972; Gey, 1924; Ehrich *et al.*, 1944). The lesions resemble those caused by severe hypoxia (Marek and Piejko, 1972) which carbon monoxide may produce by two mechanisms: (1) by replacing the oxygen in oxyhaemoglobin, and (2) by decreasing the dissociation of oxyhaemoglobin in the tissues (Ayres, Gianelli, and Mueller, 1970; Goldsmith and Landaw, 1968; Hayes and Hall, 1964).

Electrocardiographic abnormalities are often seen after carbon monoxide poisoning (Hayes and Hall, 1964; Anderson, Allensworth, and DeGroot, 1967; Leinoff, 1942; Hadley, 1952; Cosby and Bergeron, 1963; Middleton, Ashby, and Clark, 1961; Meigs and Hughes, 1952; Stearns, Drinker, and Shaughnessy, 1938; Colvin, 1928). These are usually transitory but may persist for days or weeks (Cosby

and Bergeron, 1963; Middleton *et al.*, 1961). The most common changes are sinus tachycardia, T wave flattening or inversion, and ST segment depression (Ehrich *et al.*, 1944; Cosby and Bergeron, 1963; Meigs and Hughes, 1952; Stearns *et al.*, 1938). Premature ventricular complexes (Meigs and Hughes, 1952; Stearns *et al.*, 1938), atrial fibrillation (Cosby and Bergeron, 1963; Meigs and Hughes, 1952; Stearns *et al.*, 1938), and low voltage (Stearns *et al.*, 1938) occur less frequently. ST segment elevation has occasionally been recorded (Hayes and Hall, 1964; Leinoff, 1942; Stearns *et al.*, 1938) as have conduction abnormalities (Stearns *et al.*, 1938; Colvin, 1928).

This report describes the echocardiographic findings in 5 patients who were poisoned on the same day by carbon monoxide from a defective water heater in an apartment building. The gas reached the apartments through the air conditioning ducts. The duration of the exposure is unknown. All 5 patients had been in good health before the accident.

Methods

Each patient had at least one echocardiogram during his time in the hospital. Follow-up echocardiograms were obtained 2 to 10 months after discharge.

Echocardiography was performed with a Smith Kline echograph and either a Honeywell No. 1856

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or an Electronics for Medicine DR8 strip chart recorder. The ultrasound transducer was 1.27 cm in diameter and collimated at 7.5 cm.

A sector scan of the left ventricle from base to apex (Feigenbaum, 1972) was made in each examination and the following measurements were made where echoes were simultaneously recorded from the left side of the interventricular septum, the

posterior left ventricular endocardium, and either the posterior leaflet or the chordae tendineae of the mitral valve: (1) the left ventricular internal dimension at end-diastole (LVIDd); (2) amplitude of motion of the left septal echo during ejection (LSa); and (3) amplitude of motion of the posterior left ventricular endocardial echo. Pattern of motion of the mitral valve and of the interventricular

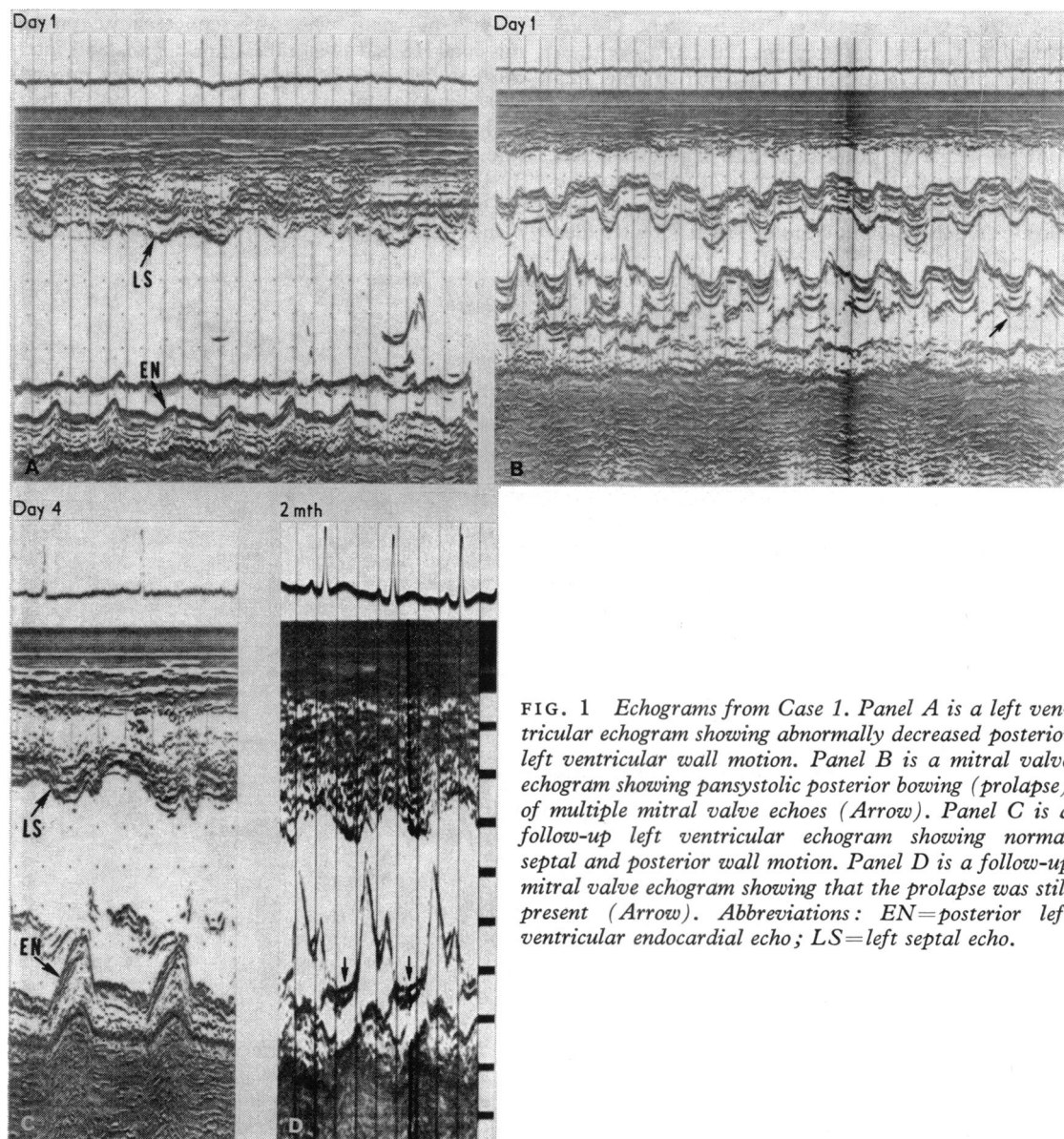


FIG. 1 Echograms from Case 1. Panel A is a left ventricular echogram showing abnormally decreased posterior left ventricular wall motion. Panel B is a mitral valve echogram showing pansystolic posterior bowing (prolapse) of multiple mitral valve echoes (Arrow). Panel C is a follow-up left ventricular echogram showing normal septal and posterior wall motion. Panel D is a follow-up mitral valve echogram showing that the prolapse was still present (Arrow). Abbreviations: EN=posterior left ventricular endocardial echo; LS=left septal echo.

septum and posterior left ventricular wall near the apex was also noted. Patients were not examined by the authors and phonocardiograms were not obtained.

Case reports

Case 1

This 16-year-old man was comatose on admission with a heart rate of 130 beats a minute, a respiratory rate of 26 cycles a minute, a blood pressure of 100/70 mmHg (13.3/9.3 kPa), and body temperature of 37.8°C. While receiving 100 per cent O₂ his arterial PO₂ was 150 mmHg (20.0 kPa) with a PCO₂ of 26 mmHg (3.5 kPa) and a pH of 7.24. His carbon monoxide level was 25 per cent on admission.

At the time of admission, his echocardiogram showed abnormally low amplitude of motion of the posterior endocardial echo and prolapse of the mitral valve throughout systole. The left ventricular

internal dimension was 5.6 cm. These findings are illustrated in Fig. 1A and Fig. 1B. A repeat echocardiogram on day four showed normal septal and posterior left ventricular wall motion (Fig. 1C). A follow-up echo two months later was normal except for the mitral valve prolapse (Fig. 1D). The left ventricular internal dimension at end-diastole on that tracing was 4.7 cm.

Case 2

The brother of the patient in Case 1 was a 20-year-old man who was semicomatose on admission. He had bilateral Babinski signs and intermittent decerebrate posturing. His heart rate was 120 beats per minute and regular, blood pressure was 170/100 mmHg (22.6/13.3 kPa), respiratory rate was 40 per minute, and temperature was 39.4°C. The carbon monoxide level was 23 per cent.

On day two he could respond to simple commands; and heart rate, respiration rate, and blood

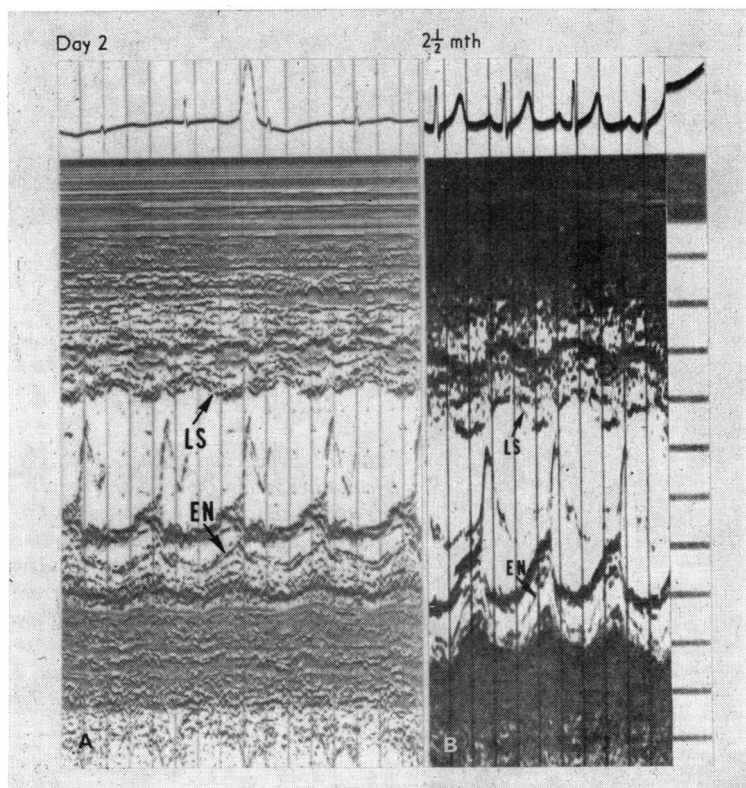


FIG. 2 Left ventricular echograms from Case 3. In panel A the posterior left ventricular wall motion is abnormally decreased. Panel B is a follow-up echogram showing that the motion did return to normal. Abbreviations: EN=posterior left ventricular endocardial echo; LS=left septal echo.

pressure were normal. The echocardiogram on day two showed posterior bowing of multiple mitral valve echoes during systole. On day four he was alert and had no complaints. An echocardiogram on day four showed slight increase in the left septal and posterior left ventricular wall motion as compared with the first tracing. An echocardiogram 3½ months later was unchanged.

Case 3

This 20-year-old woman was stuporous on admission. Her heart rate was 132 beats per minute, blood pressure was 130/90 mmHg (17.3/12.0 kPa), respiratory rate was 30 per minute, and temperature was 37°C. On 100 per cent O₂ by mask, her arterial PO₂ was 108 mmHg (14.4 kPa). The carbon monoxide level was 17 per cent. On day two she was on room air and the arterial PO₂ was 82 mmHg (10.9 kPa). Heart rate was 76 beats per minute. Her echocardiogram showed a normal size left ventricle and normal mitral valve motion. The amplitude of motion of the posterior left ventricular endocardium was less than normal as shown in Fig. 2A. She was released on day three.

An echocardiogram 2½ months later showed normal left ventricular size and wall motion (Fig. 2B) and mid-systolic prolapse of the mitral valve.

Case 4

A 24-year-old woman had been unconscious when found but was only confused on admission. She complained of dizziness and mild headache. Her heart rate was 128 beats per minute, respiratory rate was 24 per minute, blood pressure was 120/80 mmHg (16.0/10.6 kPa), and temperature was 37.6°C. On 100 per cent O₂ by mask, arterial PO₂

was 175 mmHg (23.3 kPa). The carbon monoxide level was 19 per cent.

Her echocardiogram was normal though the amplitude of the posterior endocardial echo motion was at the lower limit of normal.

On day three she was asymptomatic and was discharged from the hospital.

A follow-up echocardiogram 2½ months later was normal.

Case 5

A 29-year-old man was disorientated and aggressive on admission. His heart rate was 140 beats per minute and regular respiratory rate was 36 per minute, blood pressure was 154/95 mmHg (20.5/12.6 kPa), and temperature was 38.5°C. The carbon monoxide level was 16 per cent. After one and a half hours of 100 per cent O₂ therapy by mask, he was orientated and co-operative and his arterial PO₂ was 250 mmHg (33.3 kPa).

The first echocardiogram on this patient was taken on day seven and showed a normal LVIDd, decreased septal motion at the base (Fig. 3A), with paradoxical septal motion near the apex (Fig. 3B). The amplitude of the left ventricular posterior wall motion was at the upper limits of normal.

An echocardiogram obtained 10 months later continued to show abnormal septal motion.

The echocardiographic findings for all 5 patients are summarized in the Table.

Discussion

After acute carbon monoxide poisoning, 4 of the 5 patients had echocardiographic abnormalities. While these observations do not establish that

TABLE Echocardiographic findings

Case No.	Hospital day and follow-up	LVIDd (cm)	LSa (cm) (N=0.3-0.8 cm)	ENa (cm) (N=0.8-1.6 cm)	Mitral valve
1	1	5.6	0.4	0.6	Prolapse
	4	5.6	0.6	1.7	Prolapse
	2 mth	4.7	0.7	1.4	Prolapse
2	2	4.8	0.6	1.2	Prolapse
	4	4.8	0.8	1.3	Prolapse
	3½ mth	4.5	0.7	1.3	Prolapse
3	2	4.4	0.3	0.6	Normal
	2½ mth	4.6	0.6	1.3	Prolapse
4	2	4.5	0.6	0.9	Normal
	2½ mth	4.7	0.6	1.0	Normal
5	7	4.7	0	1.6	Normal
	10 mth	4.7	0	1.4	Normal

LVIDd=left ventricular internal dimension at end-diastole; LSa=amplitude of left septal motion during ejection; ENa=amplitude of posterior left ventricular endocardial echo motion.

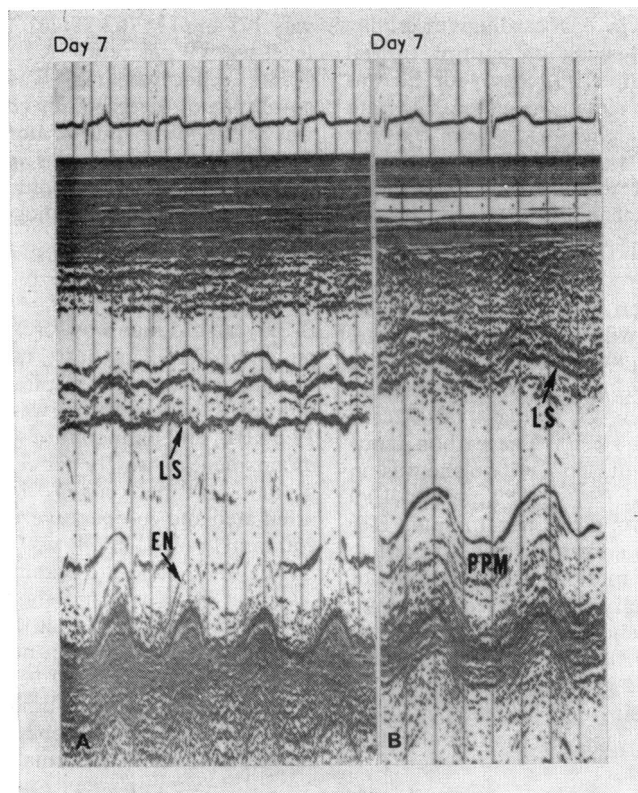


FIG. 3 Left ventricular echograms from Case 5. Panel A shows absence of left septal echo motion during ejection near the base of the ventricle. Panel B shows paradoxical left septal echo motion in an area at the level of the posterior papillary muscle (PPM). Abbreviations: EN=posterior left ventricular endocardial echo; LS=left septal echo.

carbon monoxide was the cause of the abnormalities or that its mechanism of action was myocardial oxygen deprivation, pre-existing cardiac disease is unlikely since all 5 patients were previously in good health and under 30 years of age.

Echocardiograms

Left ventricular internal dimension The left ventricular internal dimension at end-diastole (LVIDD) was normal in every echocardiogram when the body surface area was considered. In Case 1 the LVIDD decreased from 5.6 cm on the first hospital day to 4.7 cm on a follow-up tracing two months later. While this is a significant change and suggestive of improved left ventricular function, both values are normal and the difference could be the result of physiological variation.

Echocardiographic ejection fractions were not calculated in these cases since their reliability depends on symmetrical contraction and, at least

in Case 5, segmental abnormality of contraction was present with the septal motion paradoxical and with the amplitude of posterior left ventricular wall motion at the upper limit of normal.

Left ventricular wall motion Abnormalities of left ventricular echo motion have been shown in patients with cardiomyopathy (Corya *et al.*, 1974; McDonald, 1973) and in patients with obstructive coronary artery disease (Jacobs *et al.*, 1973; Corya *et al.*, 1975; Ratshin, Rackley, and Russell, 1972; Heikkilä and Nieminen, 1975). It may occur transiently during spontaneous or stress induced angina pectoris (Jacobs *et al.*, 1973; Widlansky *et al.*, 1975). Though the finding is non-specific, it probably is the result of myocardial hypoxia in these cases of carbon monoxide poisoning. In Cases 1 and 3 the motion returned to normal when the echocardiogram was repeated. In Case 5 the abnormal septal motion was still present 10 months later.

Mitral valve prolapse

There have been numerous reports showing excellent correlation between echocardiographic and angiographic findings of mitral valve prolapse in patients with a variety of auscultatory and phonocardiographic findings (Dillon *et al.*, 1971; Popp *et al.*, 1974; DeMaria *et al.*, 1974). The posterior motion of the mitral valve leaflets may begin very early in systole, giving the echo a bowed or hammock appearance, or it may begin abruptly in mid-systole (Popp *et al.*, 1974; DeMaria *et al.*, 1974; Shah and Gramiak, 1970). Mitral valve prolapse has become a common echocardiographic finding (Popp *et al.*, 1974; DeMaria *et al.*, 1974) and both the aetiology and the clinical significance of it are undetermined. Because previous echocardiograms were not available it is uncertain that carbon monoxide was responsible for prolapse in our three cases. None of the patients had physical findings suggesting Marfan's syndrome or other congenital abnormalities. Since 2 of the 3 patients were brothers, a familial disorder cannot be ruled out as the aetiology in those cases. However, in view of the known frequency of papillary muscle lesions in patients who die with carbon monoxide poisoning (Marek and Piejko, 1972; Gey, 1924), papillary muscle dysfunction must be considered a likely cause of the prolapse in these cases.

Conclusion

These echocardiographic observations suggest that myocardial injury may occur in non-fatal cases of carbon monoxide poisoning. The damage, as shown by abnormal left ventricular echo motion, is reversible in some cases; and mitral valve prolapse may result from papillary muscle involvement.

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